CASE REPORT

PRIMARY PYLORODUODENAL TUBERCULOSIS PRESENTING AS GASTRIC OUTLET OBSTRUCTION

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ABSTRACT

A 45-year-old female patient from Jimma town was admitted to Jimma university teaching hospital on November 2002 for chief complaint of severe vomiting of ingested material that failed to respond to the routine antacid medications. The patient was diagnosed as a case of gastric outlet obstruction secondary to primary pyloroduodenal tuberculosis. The overall clinical presentations as well as laboratory and histopathology reports are discussed. [Ethiop J Health Sci 2003; 13(2):131-133]

INTRODUCTION

Intestinal tuberculosis comprises of about 5% of all cases of tuberculosis in susceptible communities. Intestinal tuberculosis establishes itself in the lymphoid follicles of the Peyer’s patches and spreads to the submucosal and subserosal planes. The resulting chronic inflammation causes much thickening of the intestinal wall and consequent narrowing of its lumen with an early involvement of regional lymphnodes, which may later caseate (1-3). Tuberculosis of the stomach and duodenum is rare in patients with pulmonary tuberculosis and primary involvement of tissues is even rare (4).

Primary gastroduodenal tuberculosis is exceedingly uncommon condition and clinically it can mimic other gastrointestinal disorders. The aim of this report is, therefore, to alert the health professionals for the ever-increasing trends of extra pulmonary tuberculosis in this era of immunodeficiency states.

CASE REPORT

M.G., a 45-year-old female patient from Jimma town, Southwestern Ethiopia was admitted to surgical ward, Jimma university teaching hospital on November, 2002 for complaints of severe vomiting of ingested materials that persisted for more than two months duration. Prior to admission she had several visits to the hospital for recurrent epigastric burning sensation where she was repeatedly prescribed with different antacid suspensions, which did not result in pain relief. The patient had no history or physical evidence of pulmonary tuberculosis or other systemic disorders, except a recent bout of diarrhea. Thus a working diagnosis of gastric outlet obstruction secondary to chronic duodenal ulcer was made. The patient was investigated and prepared for operation.

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Relevant physical examinations were registered. Patient was very emaciated with stable vital signs and scaphoid abdomen, which moved with respiration. There was no fever and no area of abdominal tenderness. Succession splash was noted, and normoreactive bowel sounds heard.

**Laboratory data:**
Urine analysis: Albumin: 2++, Sugar: negative, RBC = 8-10 / Hpf, WBC = 3-5 /Hpf
Stool: Entameba histolytica and Giardia lamblia trophozoites were recovered.

**Radiology report:**
Barium meal revealed complete obstruction of the second part of the duodenum resulting in extensive dilatation of the stomach and proximal part of duodenum.

**Operation remarks:**
An ill-defined pyloric mass was identified during operative procedure with peritoneal reaction. Subsequently an incisional biopsy was taken from the area of maximal nodularity without undertaking major surgical procedures.

**Histopathologic procedure:**
The specimen was kept within well-formalized, labeled container and sent to the Pathology Department. On macroscopic examination, the specimen revealed a gray white firm tissue piece 0.5 cm in diameter that was subsequently underwent successive histopathologic procedures including dehydration with inter-grades alcohol, cleaning with xylenes, impregnated with paraffin wax and then cutting with microtome to 0.5 micron thick sections and finally it was stained with Hematoxylin & Eosin stains and visualized under light microscope.

**Histopathology report:**
The histopathologic sections showed aggregates of epitheloid cells granulomas, Langhan’s giant cells as well as areas of caseous necrosis along with few glands. The features were typical of tuberculosis. Tissue sections for Acid-fast bacilli was not done since it presented the most florid microscopic presentation and a screening tests for HIV, infection was not undertaken.

**DISCUSSION**
Gastric outlet obstruction is obstruction of the stomach at pyloroduodenal area and is caused by cicatrization and edema of the recurrent duodenal ulcer. Gastric outlet obstruction is a common and early complication of duodenal ulcer in Africa (2). However gastric outlet obstruction due to other causes is uncommon and this report discusses tuberculosis as a cause of gastric outlet obstruction involving the pyloroduodenum without clinical or otherwise evidence of pulmonary tuberculosis as were repeatedly reported by Tromba et al (6) and Sing et al (7).

Thus, this report with no other manifestations of pulmonary tuberculosis or other tuberculous lesions, was designated as primary lesion that is much uncommon than secondary gastroduodenal tuberculosis (4). Gastroduodenal tuberculosis with gastric obstruction is however commonly encountered in those patients with concomitant pulmonary tuberculosis (5). The clinical presentation of gastric outlet obstruction are nonspecific and therefore confusing as various case reports depict (1,4, 6) and this profile was also surfaced in our case where our patient was persistently managed till the pathology report as a case of gastric outlet obstruction secondary to chronic peptic ulcer disease. Similarly, Wouclstram et al (2) disclosed that gastroduodenal tuberculosis is uncommon condition that can mimic other gastrointestinal disorders. It usually occurs secondary to pulmonary tuberculosis. All clinicopathologic profiles of this patient could hint much poorer immunosurveillance that might be precipitated by immunodeficiency states where HIV/AIDS and under nutrition are so rampant.
In conclusion, this case report can cast a further hint to health professional for the ever-diversifying trends in extra pulmonary tuberculosis in this era of immunodeficiency states.

ACKNOWLEDGMENT

We extend our heart-felt gratitude to Dr. Tigistu Adamu from Surgical Department in Jimma University for his invaluable assistance while we were writing this report.

REFERENCES